Dissection of the abdominal aorta in blunt trauma: Endovascular or conventional surgical management?

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Background: **Dissection of the abdominal aorta caused by blunt trauma is a rare injury, often complicated by thrombosis within the true and false lumens and sometimes aortic rupture. The mortality rate with conservative medical management is approximately 75%, whereas it ranges from 18% to 37% with surgical treatment.**
Methods: **At our institution, 7 of the 87 patients admitted because of blunt aortic trauma, between January 1995 and January 2002, had abdominal aortic dissection.**
Results: **Four patients were treated using endovascular techniques by percutaneous stent placement. The indications for endovascular management were lower limb ischemia in one case and extension of the dissection in two; one patient was asymptomatic. Aortic dissection was complicated by ischemic paraplegia in two patients, and both were treated by conventional operative repair. One patient was managed medically because of a minimal intimal disruption.**
No deaths were related to the aortic dissection or its treatment. Angiographic and computed tomographic (CT) studies showed thrombosis of the false lumen and complete obliteration of the dissection in the endovascular group. The neurologic condition of the two paraplegic patients either cleared completely or partially improved.
Conclusion: **In the absence of ischemic paraplegia or other injuries that require emergency surgery, endovascular treatment is a safe and efficient method for treating traumatic infrarenal aortic dissection. (J Vasc Surg 2003;38:997-1004.)**

Nonpenetrating injuries of the abdominal aorta are rare, and only 25% of them result in aortic dissection.[1] Because the aorta is highly protected by virtue of its anatomical location, aortic dissection is usually associated with major trauma and damage to other structures. Surgical repair is, for the most part, considered as the preferred treatment because the problem is often fatal. Lock et al[2] reported 75% mortality in patients treated medically.

In a previous report,[3] we described one patient with traumatic infrarenal aortic dissection who was successfully treated by percutaneous placement of endovascular stents. This series deals with six other cases of infrarenal aorta traumatic dissection secondary to blunt abdominal injuries. The aim of this study is to discuss the management of infrarenal aortic dissection in blunt trauma with special emphasis on clinical and radiologic findings, as well as indications and modality of surgical treatment.

MATERIAL AND METHODS

This study includes 87 patients with blunt aortic trauma admitted to our institution between January 1995 and January 2002. Seven cases of traumatic dissection of the infrarenal aorta were found, accounting for 8% of the total patients. Blunt trauma of the descending thoracic aorta occurred in 81% of patients, whereas 11% of patients had other blunt injuries of the abdominal aorta (false aneurysm, intramural hematoma).

On their admission, all seven patients underwent a physical examination, a laboratory evaluation, an abdominal radiograph, and a chest radiograph. A thoracic CT scan was obtained in all but one patient, in whom the physical examination and chest radiograph failed to disclose any evidence of an intrathoracic lesion. An abdominal CT scan with intravenous contrast was employed in all patients to establish the diagnosis. A multiple-view angiography was performed with a brachial approach to document the extent of the dissection and to rule out the presence of major visceral branch involvement. Angiography was not performed in patient 6 because his vital signs were unstable, necessitating emergency surgery. A magnetic resonance (MR) study, which focused on the spinal cord, was performed within the first 2 hours after admission in the two patients with paraplegia (patients 6 and 7).

Angiography and CT scan were performed before hospital discharge. Surveillance including physical examination, Doppler ultrasound, and CT scan were scheduled at 1, 3, and 6 months and annually thereafter.

RESULTS

The mean age of patients at the time of diagnosis was 46.4 years (range, 18-89 years). The 89-year-old patient was the only woman of this report (patient 2).

Road accidents ranked first as the cause of trauma. One patient had a seat belt injury (patient 3), and two were struck by an unknown structure after being ejected from

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Fig 1. The only patient (patient 5) who was not treated surgically had a limited juxtarenal dissection, which began immediately below the right renal artery and extended 2 cm distally on the right side of the aorta.

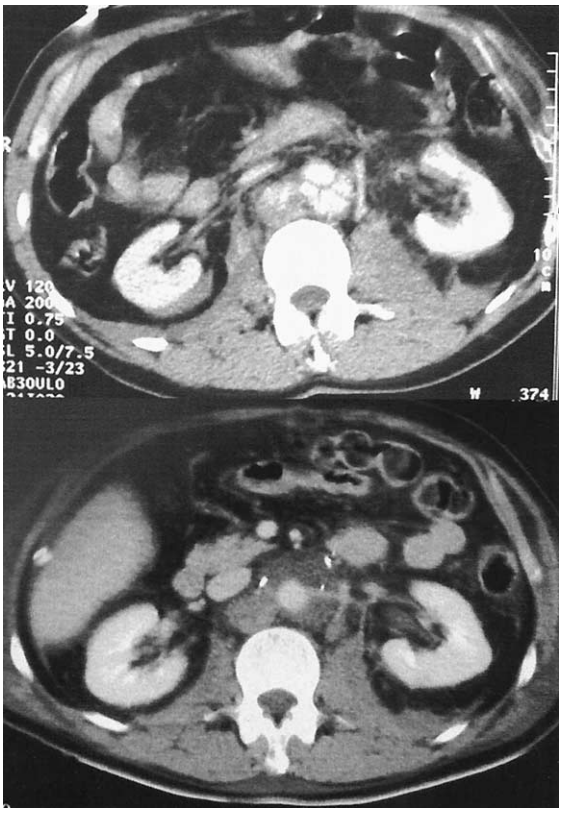

Fig 2. The preoperative CT scan from the sixth patient with infrarenal aortic dissection, splenic rupture, retroperitoneal hematoma, and left phrenic disruption. The immediate postoperative CT scan confirms the quality of the revascularization.

their car (patients 6 and 7). Four patients had blunt injury of the abdominal aorta from miscellaneous causes: one fell on an enormous piece of ceramic pottery (patient 2), one fell from stairs and struck a large table with his abdomen (patient 4), one was crushed against a wall by a fork-lift truck (patient 1), and one was hit by a horse-shoe (patient 5).

The time from injury to hospital admission ranged from 1 to 12 hours after the initial injury. The clinical presentation was pain and findings of an acute abdomen in all cases. All patients in this series were assessed and stabilized in the admitting area of the neurosurgical or general trauma center. Findings at presentation were hypovolemic shock, which responded well to fluid administration in five cases (patients 1, 3, and 5-7); paraplegia in two patients (patients 6 and 7); lower limb ischemia in two patients (patients 2 and 6); and isolated back "knife-like" pain and acute abdomen in one patient (patient 4). Other presenting symptoms included respiratory distress (patients 1, 6, and 7), hematoma of the abdominal wall (patients 2-4), and right flail chest (patient 7). Clinical findings, symptomatology, and associated injuries are summarized in Table I.

Paraplegia occurred in the first hour after the initial trauma. It was associated with lower limb ischemia in the sixth patient and with acute abdominal findings, respiratory distress, and shock in both patients. No direct traumatic injury, such as echymoses or hematoma, was found in the area of the vertebral column in those two cases. Patients with paraplegia were initially admitted to the neurosurgical emergency unit.

An "immediate diagnosis" (discovery of the injury within 24 hours of occurrence[2]) was made in five cases (patients 1, 2, and 5-7) and an "early diagnosis" (diagnosis at 1-7 days[2]) in two cases (patients 3 and 4).

The delay between admission to the hospital and vascular surgical management ranged from 120 minutes (patient 6) to 4 months (patient 3) (Table II). Three patients (patients 2, 6, and 7) were operated on within 24 hours because of an ischemic complication of the dissection (patients 6 and 7 were operated on immediately). Three patients (patients 1, 3, and 4) underwent a delayed treatment because spontaneous thrombosis of the false lumen was expected or because of their associated lesions. The third patient (patient 3) was transferred to the operative room for surgical exploration because of a massive hemoperitoneum diagnosed by ultrasound. Bleeding from a mesenteric vessel was controlled, and a segment of small intestine was removed. Immediately after the surgical procedure, a leg

Table I. Data on 7 patients with infrarenal aortic traumatic dissection after blunt trauma

No.	*Age*	*Sex*	*Causes*	*Clinical presentation*	*Associated lesions*	*Lesion*	*Site*
One	34	M	Crushing injury	Shock Abdominal pain Epigastric ecchymoses	Pancreatic contusion Retroperitoneal Hm	Aortic dissection	Below IMA
Two	89	F	Fall	Acute right limb ischemia Abdominal pain	Abdominal wall Hm Retroperitoneal Hm	Aortic and left iliac dissection, right iliac occlusion	Below IMA
Three	41	M	Road accident	Collapse Acute abdomen	Mesenteric Hm Retroperitoneal Hm	Aortic and bilateral iliac dissection	Level of IMA
Four	54	M	Fall	Abdominal and back pain Epigastric ecchymoses	Abdominal wall Hm Retroperitoneal Hm	Aortic and left iliac dissection	Below RA
Five	18	M	Horse shoe	Shock Acute abdomen Epigastric echymoses	Gastric disruption Retroperitoneal Hm	Aortic dissection	Below right RA
Six	41	M	Road accident	Shock Paraplegia Acute right limb ischemia Respiratory distress	Spinal chord ischemia Left flail chest Left phrenic disruption Splenic rupture Retroperitoneal Hm	Aortic and iliac dissection Aortic thrombosis	Below RA
Seven	48	M	Road accident	Shock Paraplegia Respiratory distress	Spinal chord ischemia Right phrenic disruption Hepatic contusion Retroperitoneal Hm	Aortic dissection and false aneurysm	Between RA and IMA

Hm, Hematoma; *IMA*, inferior mesenteric artery; *RA*, renal artery.

ischemia was diagnosed. Arteriography showed an isolated dissection of the infrarenal aorta extending to both iliac arteries. The patient had femoral pulses but a compartmental syndrome leading to fasciotomies and renal insufficiency requiring hemodialysis. As there was no sign of spontaneous thrombosis of the false lumen after 4 months, the decision was made to treat the dissection.

RADIOLOGIC FINDINGS

Although both CT scans with intravenous contrast and angiography were used, CT scans established a definite diagnosis in five patients (patients 1, 3, 4, 6, and 7) and provided suspicious findings in the other two patients. In case of endovascular treatment, CT scan allowed precise measurement of the aortic lumen diameter to ensure that the chosen stent would fit the aortic wall. The main associated lesions were also diagnosed by CT scan.

The diagnosis was made by angiography alone in two cases (patients 2 and 5) (Fig 1). The intimal flap of the dissection was identified below or at the level of the inferior mesenteric artery (IMA) in three cases. In three other cases, angiography demonstrated an aortic tear between the origins of the renal arteries and the IMA. In one case (patient 5), an angiogram revealed that the proximal extension of the dissection was close to the right renal artery, but the vessel was patent.

Paraplegia caused by spinal cord ischemia as the first sign of the traumatic infrarenal aortic dissection was noted in two patients (patients 6 and 7) (Fig 2). While they were in the Neurosurgery Department, the anomaly was diagnosed after dorsal and lumbar MR. Imaging was performed 1 to 2 hours after injury. MR was useful in assessing the absence of skeletal and/or ligamentous injuries and spinal cord compression. MR detected an ischemic zone in the spinal cord approximately from the level of the second to the fourth lumbar vertebral body.

TREATMENT

The two patients with spinal cord ischemia (patients 6 and 7) underwent graft interposition and revascularization of the lumbar arteries as quickly as possible.

The sixth patient underwent a trifurcated antimicrobial InterGard Silver (diameter 18/9 mm; Intervascular SAS, La Ciotat, France) aortoaortic and bifemoral aortic replacement (Fig 3). This prosthesis was chosen because of an uncertain "infection free" surgical area induced by associated lesions (large bowel serrosa injury). In the patient with juxtarenal extension of the dissection, the suprarenal aorta was temporarily occluded to permit reinforcement of the aorta with biological glue and polytetraflouroethylene bolsters. Subsequently, the aortic portion of a bifurcation graft was anastomosed end-to-end to the infrarenal aorta. The third and fourth lumbar arteries were revascularized by a large distal end-to-end anastomoses. Limbs of the aortic graft were attached to the femoral arteries because of bilateral external iliac artery occlusion. Management of associated injuries involved closure of a diaphragmatic rupture, splenectomy, and large bowel closure. The last patient was managed by an aortoaortic bypass with proximal and distal end-to-end anastomosis. In this case, it was possible to clamp the infrarenal aorta proximal to the aortic dissection. The distal extension of the anterior dissecting process was managed by an occlusion of the false lumen with glue and polytetrafluoroethylene patch. The prosthesis was an Inter-

Table II. Management of patients with infrarenal aortic traumatic dissection after blunt trauma

No.	*Delay**	*Management*	*Device*	*Results*	*Follow-up*
One	9 d	Endovascular	Two Wallstents Aorta: R09F16030† Left iliac: R07F09043	Thrombosis of the false lumen	Alive
Two	1 d	Endovascular	Four Wallstents Aorta: R09F16037 Right iliac: R07F10031, R09F14030 Left iliac: R09F16037	Thrombosis of the false lumen	MI at 3 years
Three	4 mo	Endovascular	Palmaz P308 (aorta)	Thrombosis of the false lumen	Alive
Four	15 d	Endovascular	Two Palmaz (XXL aortic) One Wallstent Left iliac: R09F14030	Thrombosis of the false lumen	Alive
Five		Medical conservative		Stability of the false lumen	Alive
Six	2 h	Surgical conventionnal	Trifurcated aortoaortic and bifemoral graft (Dacron) Lumbar arteries revascularisation	Lumbar, hypogastric and femoral arteries revascularisation	Alive Right limb neurologic impotency
Seven	3 h	Surgical conventionnal	Aortoaortic tube graft Lumbar arteries revascularisation	Lumbar, iliac arteries revascularisation	Alive

MI, Myocardial infarction.

*Delay: Delay between admission on the hospital and surgical management.

†The reference R09F16030 indicates that the stent must be placed using a 9 F introducer and that the diameter is 16 mm and length 30 mm at full unfettered deployment.

Gard Silver (diameter 18 mm). The third, fourth, and fifth pairs of lumbar arteries were reimplanted in a large distal anastomosis. The phrenic disruption and hepatic contusion were managed at the same time.

Endovascular management was performed in four cases. In asymptomatic patients, the absence of delayed spontaneous thrombosis of the dissection without anticoagulation led to treatment. All endovascular procedures were performed in an operating suite with a mobile C-arm (Sire-mobil, Siemens, Munich, Germany). Patients were prepared and draped for femoral arteriotomy and potential emergency transperitoneal aortic approach, and they were fully monitored.

The first patient presented with an aortic dissection that began above a patent inferior mesenteric artery and extended distally to the left common iliac artery. Using a left 9F femoral approach over a 0.035-inch Terumo guidewire (Terumo Medical Corporation, Tokyo, Japan), a Wallstent (Schneider Wallstent, Pfizer, Minneapolis, Minn) was placed into the aorta. A second Wallstent was placed in the left common iliac artery. An angiographic examination demonstrated obliteration of the dissection except at the aortoiliac junction where the stent was displaced. Contrast study of the IMA was normal. Angiographic and CT scan performed 10 days later demonstrated complete obliteration of the false lumen, including the uncovered segment between the two stents.

The aortic dissection in the second patient began distal to the inferior mesenteric artery, occluded the right common iliac artery, and involved both external iliac arteries. The patient was treated by placement of four Wallstents, one in the aorta, two in the right iliac artery, and one in the left iliac artery. Angiography and CT scans performed 8 days later showed satisfactory obliteration of the dissection.

A persistent dissection of the infrarenal aorta 4 months after the accident led us to treat the third patient by an endovascular procedure. A Palmaz (Palmaz stent, Johnson & Johnson Interventional system, Warren, NJ) balloon-expandable stent (P 308) was deployed above the aortic bifurcation using a 15-mm diameter balloon. Arteriography and CT scan after the procedure confirmed a successful management.

An infrarenal aortic dissection extending to the left main iliac artery was identified in the fourth patient. The patient was treated by placement of two Palmaz balloon-expandable stents (XXL) using a 20-mm–diameter balloon in the aorta and by placement of one Wallstent in the left iliac artery. The first aortic stent was placed 5 mm below the renal arteries and the second immediately above the aortic bifurcation. The postprocedure angiogram and CT scan showed the complete resolution of the dissection.

The only patient (patient 5) who was not treated surgically had a limited juxtarenal dissection, which began immediately below the right renal artery and extended 2 cm distally in the right side of the aorta (Fig 1). A gastric disruption necessitated an emergency operation. As there was no sign of extension of the limited dissection noted on the postoperative radiologic examination, the patient was managed conservatively.

POSTOPERATIVE PERIOD AND FOLLOW-UP

Because of the associated lesions, anticoagulation was avoided in all cases in the postoperative period. In the endovascular group, there were no vascular complications

Fig 3. The sixth patient underwent a trifurcated antimicrobial InterGard Silver (diameter 18/9 mm) aortoaortic and bifemoral aortic replacement. The third and fourth lumbar arteries were revascularized by a large distal end-to-end anastomoses. Limbs of the aortic graft were attached to the femoral arteries because of bilateral external iliac artery occlusion.

or deaths. Patients were released from our surgical department 2 to 13 days after treatment. Three of the four patients recovered completely. One patient (patient 3), however, developed an anterior compartmental syndrome with foot drop and required physical therapy. All patients were followed up with physical examination, ultrasonography, and CT scans for a period ranging from 25 to 90 months. One patient, an 89-year-old woman with documented coronary artery disease, had a fatal myocardial infarction 32 months after treatment of her aortic dissection. In the other cases, physical examinations and imaging surveillance confirmed complete obliteration of false channels. All stents appeared to be positioned satisfactorily, and all hematomas were resolved. During follow-up, none of the patients were found to have aortic enlargement on CT scans.

In the conventional surgical group, both paraplegic patients exhibited an incomplete paraparesis postoperatively. In one, all neurologic symptoms promptly cleared, whereas the other patient improved slowly but developed a partial palsy of the right lower extremity that still requires physical therapy 7 months after aortic trauma.

During follow-up, an angiography was performed in both cases, showing an antegrade perfusion of the lumbar arteries. In both patients, CT scans did not disclose any signs of graft infection.

DISCUSSION

The exact incidence of infrarenal aortic dissection in blunt trauma is unknown, because many patients with intimal disruption do not reach the trauma center alive. Killen[4] has reported only one aortic wall disruption out of 1320 patients with blunt abdominal trauma.

Two major mechanisms have been proposed to explain infrarenal aortic dissection during nonpenetrating trauma: direct from the blunt trauma with aortic compression against bony structures of the spine, especially in seatbelt injuries,[6-8] and indirect as a result of deceleration force, especially in high-speed collisions.[9] Atheromatous aortic plaques are believed to be a possible contributing factor to abdominal aortic dissection because less force is required to injure the intima over a plaque in a rigid atheromatous aorta.[10-12] It may explain why this lesion is far from being rare in older patients.

The various clinical presentations have been previously explained.[2,12-14] Most cases of blunt injury of the abdominal aorta presented no diagnostic problems. Nevertheless, a high rate (34.3%)[15] of delayed diagnosis is reported in the literature, related to a hidden vascular symptomatology (52%) or to the emergency-associated visceral lesions. A precise knowledge of the circumstances of the trauma as well as a high suspicion index of this condition should routinely lead to a CT scan and an aortic angiography.

In patients with spontaneous abdominal aortic dissection, Graham[16] found that the combined use of real-time abdominal ultrasonography and Doppler flow studies was an effective means for demonstrating intimal flaps as well as evaluating blood flow in both false and true lumens. In our series, such modalities were unable to assess the diagnosis, to evaluate the proximal and distal limits of the dissection, and to determine the associated involvement of visceral arteries, probably because of retroperitoneal hematoma and the associated lesions. Moreover, this examination is frequently unable to show short intimal flaps[17,18] and requires well-trained operators that are not always available in emergency.

Contrast-enhanced CT scan and angiography have been currently performed to establish diagnosis in most of the series. An associated careful screening of the thoracic aorta to eliminate any dissection at this level should be performed.[18] CT scan of the abdomen usually shows direct signs such as intraluminal flap on successive images, double channel,[19] or indirect signs such as retroperitoneal hematoma or abnormal opacification of the aortic lumen. CT scans may also disclose complications of dissection such as aortic thrombosis, false aneurysms, and aortic rupture. CT is able to provide complementary data such as precise vessel diameter and associated lesions that guide surgical management. Errors in interpretation may be caused by thrombosis of the false lumen or the presence of an underlying atherosclerotic abdominal aortic aneurysm.

For most authors,[19] a multiple-view angiography remains the definitive radiologic examination of choice in most acute traumatic dissection. The most common angiographic signs are the opacification of the false lumen, the direct visualization of the endoluminal flap, and smooth narrowing of the infrarenal aorta. Aortic dissection may be overlooked when the false channel is thrombosed or minimally perfused and when blood flow is present in both the true and false lumens.[20] In our series, all dissections were diagnosed by CT and angiography.

The association of paraplegia and abdominal aortic dissection constitutes a special problem. The neurologic deficit occurs immediately after aortic injury in 60% of cases.[1] The origin of peripheral neurologic signs is not reported in major series of literature but seems to be related to peripheral nerves ischemia reversible after revascularization[15] or traumatic bony or ligamentary spinal lesions. Paraplegia, occurring as a result of spinal cord ischemia, accounts for about 10% of the cases reported in the literature.[1] Several risk factors have been suggested as a cause of real ischemic paraplegia, which include possible interruption of the spinal cord blood supply via the greater medullary artery (the so-called artery of Adamkiewicz [AK]), presence of episode of major hemodynamic shock, thrombosis of the hypogastric arteries, a direct lesion of the AK, and a thrombosis of an infrarenal originating AK. In our series, a segmentary lumbar spinal cord ischemia was established by MR in two patients. Proper therapeutic management of acute spinal ischemia depended considerably on the assessment of the condition of the underlying spinal cord by MR, which could establish the absence of instability of the surrounding bony architecture and the lesion level. In this situation, this radiologic examination must be performed in emergency when the patient's hemodynamic status allows it.

The occurrence of other associated abdominal lesions in 58%[1] of the patients illustrates the importance of a complete abdominal exploration in blunt injury of the abdominal aorta. Vascular signs of dissection are often masked[20-23] by associated lesions. The incidence of small bowel disruption, large bowel disruption, and mesenteric tears is 18%, 16%, and 10%, respectively; all are potential sources of contamination and may alter surgical management. In our series, associated visceral lesions were noted in 42% of patients; however, operative contamination occurred in only one patient. Because the mechanisms of injury are similar in abdominal aortic dissection and bowel disruption, a systematic multidisciplinary evaluation of each patient is mandatory.

Appropriate management of traumatic abdominal aortic dissection is not well defined. The rare data available in the literature demonstrate a failure of the nonoperative approach to this rare condition and suggest that disease progression may be inevitable. According to Le Pimpec-Barthes and Kieffer,[1] a late diagnosis (ie, diagnosis after a period longer than a week) of an aortic injury is made in 36% of patients. In these cases, the delayed symptoms are either claudication or are related to a false aneurysm. In a review of blunt trauma to the abdominal aorta based on one case report and a review of the literature (32 cases), Lock et al[2] reported four patients who underwent nonoperative therapy; three of these patients subsequently died, and one did well. There is a general consensus that surgical treatment is indicated. Thus, we suggest that all traumatic aortic injuries should be treated either by endovascular stenting, if feasible, or by conventional prosthetic graft replacement in selected patients. In the fifth patient, the dissection began immediately below the right renal artery and extended 2 cm distally in the right side of the aorta. An endovascular treatment could lead to an occlusion of the left renal artery, as shown in Fig 1. As the natural history of minor intimal tears is unknown, we elected not to treat this patient in order to avoid the possibility of renal artery thrombosis.

In comparison with spontaneous abdominal aortic dissection, the surgical mortality is worse in traumatic cases because of associated retroperitoneal venous injury and bacterial contamination.[25,26] Two authors[1,26] recommend initial clamping of the supraceliac aorta to avoid initial life-threatening hemorrhage despite increased risk of paraplegia. Emergency conventional surgery is recommended for the following special circumstances: medically uncontrolled hemodynamic shock, lower limb acute ischemia, and proven ischemic medullar paraplegia.

Conventional surgery included simple flap suture to correct a limited intimal disruption, thromboendarterectomy in case of extensive dissection,[13] or graft replacement in case of badly damaged aorta after proximal and distal closure of the dissection. Lassonde and Laurendeau[13] recommend an extra-anatomic bypass in septic acute vascular cases with thoracic or axillary proximal anastomosis. In septic cases, Kieffer et al[28] recommend an in situ allograft replacement to decrease the risk of secondary abdominal aortic rupture. In the sixth and seventh patients, we performed in situ aortic replacement with an InterGard Silver prosthesis because of its availability and its antimicrobial characteristics. In situ aortic replacement was performed to revascularize lumbar arteries and internal iliac arteries in case of spinal cord ischemia. Le Pimpec-Barthes and Kieffer,[1] in a literature review, point out that only hypovolemic or "false" paraplegia is regressive after surgery. With these two new cases, we suggest that patients with paraplegia related to spinal cord ischemia also benefit from prompt conventional surgical management.

Excluding spinal cord ischemia, medically uncontrolled hemodynamic shock, and tortuous iliac arteries, interventional angiographic techniques provide a less invasive alternative therapeutic procedure. Recent reports[3,19,29-33] mention good results in endovascular treatment of acute traumatic abdominal dissection, especially in the inferior inframesenteric segment. Nevertheless, the potential complexity of the entry–re-entry situation and the risk of aortic wall injury require the possibility of immediate laparotomy.[34]

Peterson et al[19] and Nishimura et al[31] successfully treated an aortic dissection by percutaneous balloon fenestration followed by stent placement. In both cases, lower limb ischemia was relieved and the 2-year follow-up did not reveal any abnormality. In our series, no patient underwent

endovascular fenestration. We think that isolated fenestration only treats the ischemic consequences of the dissection without treating the cause; however, it may be useful when dissection extends above the renal arteries. Stenting the dissecting process is an alternative to fenestration, and experimental studies[35,36] have demonstrated that stenting is more efficient if stents are inserted at the site of the intimal tear and cover the entire dissected lumen. In a previous report,[3] we described the first case of stented traumatic infrarenal aortic dissection. Except in selected cases, we think that endovascular treatment should be the first therapeutic option for the following main reasons. Endovascular approach may be performed immediately after closure of a laparotomy for septic injuries; this less invasive technique avoids aortic cross-clamping and retroperitoneal dissection. The limited length of dissection is a characteristic feature of traumatic aortic lesions,[30] making it easier to cover the whole dissection by stenting. In juxtarenal dissection, the risk of renal artery thrombosis may exclude endovascular management, although covering IMA ostium was uneventful in our series. We used two types of stent devices, preferring ballon-expandable stents when precise placement or high radial strength (in the third case, we speculated that fibrosis was important 4 months after the initial trauma) was required. The choice of self-expanding stents was made in the other cases because of their great flexibility, allowing conformation to the curving vessels and their permanent pressure applied on the intimal flap.

CONCLUSION

The data of the literature and our experience suggest that all traumatic aortic injuries should be treated either by stenting if feasible or by surgical replacement in selected cases. In the absence of ischemic paraplegia or other injuries that require emergency surgery, endovascular treatment is a safe and efficient method for treating traumatic infrarenal aortic dissection.

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